# Permissive Hypercapnia

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*A strategy used to avoid damage during ventilation for certain patients is permissive hypercapnia. Among candidates prone to lung damage due to high pressure and/or volume are people with ARDS and sometimes people with COPD.*

With rapid advances in critical care medicine, technology, and skill, it has become clear that certain types of patients are extremely difficult to manage and ventilate successfully. Damage to the lung brought about by using mechanical ventilation (particularly ventilation using high pressure and/or volume) is a major concern, and it contributes to the difficulty of managing these patients. Certain patients have been identified as being prone to lung damage due to high pressure and/or volume: those with adult respiratory distress syndrome (ARDS), those in status asthmaticus, and sometimes those with chronic obstructive pulmonary disease (COPD).[1] A strategy used to avoid damage during ventilation is permissive hypercapnia.

Normally, the body regulates the depth and frequency of breathing to keep the PaCO2 within a normal range of 35 to 45 mm Hg. The renal system participates in acid-base regulation by increasing or decreasing levels of bicarbonate (HCO3-). The cardiopulmonary system strives to keep the body's pH between 7.35 and 7.45. Lung disease can interfere with effective alveolar ventilation, producing a buildup of carbon dioxide and an elevated PaCO2 and resulting in respiratory acidosis. In chronically ill, stable patients, respiratory acidosis is compensated for and the pH is maintained within its normal range by the up-regulation of HCO3-, which acts as a buffer to counteract the acidosis caused by the increased PaCO2.

**Ventilator-Induced Lung Injury**

Using mechanical ventilation, clinicians can directly affect the PaCO2 by adjusting the frequency and tidal volume (VT). They usually try to keep the PaCO2 within the normal range; however, in difficult-to-ventilate patients, they have seen the damage associated with the ventilator when they try to maintain normal blood-gas values for pH and PaCO2. Barotrauma is lung damage from high pressure and volutrauma is damage due to high volume.[2] Mechanotrauma is the damage that results from repeatedly stretching the delicate lung tissue and from the cycle of recruiting collapsed alveoli by inflating and opening them on inspiration, then allowing them to deflate and collapse on expiration.[3] This combination of excessive stretching and the shear forces associated with the recruitment and derecruitment of alveoli results in ventilator-induced lung injury, which contributes to poor outcomes.

Mechanical ventilation has the potential to create dynamic hyperinflation, often referred to as intrinsic positive end-expiratory pressure (PEEP) or auto-PEEP, in patients who have a prolonged expiratory time, high minute ventilation, or early collapse of the airways.4 Thus, dynamic hyperinflation may be seen with mechanical ventilation in asthma, ARDS, and COPD. As dynamic hyperinflation occurs, trapped air increases in the lung, peak pressures creep up, and work of breathing increases. One of the strategies used to minimize the effects of both ventilator-induced lung injury and dynamic hyperinflation is to allow hypercapnia to occur.[4]

Permissive hypercapnia occurs when clinicians decrease alveolar ventilation and allow the PaCO2 to rise. This is done by avoiding delivery of high inspiratory pressures and/or large inspiratory volumes to the lung (setting a low VT and controlling peak inspiratory pressure). This approach is one of the key components of lung-protective strategy in mechanical ventilation and is used particularly with ARDS patients. Lung-protective strategy includes the use of smaller VTs, permissive hypercapnia, pressure-limited ventilation,

inverse-ratio ventilation, the best PEEP, and prone ventilation.[5] Guidelines[1] for initiating permissive hypercapnia call for allowing a gradual increase in the PaCO2, beginning at the rate of 10 mm Hg per hour and going up to a maximum allowable rise of 80 mm Hg per hour. In addition, a pH of 7.25 or more seems to be the most common target for acid-base balance. Protocols[1] call for the addition of buffering agents such as sodium bicarbonate, tromethamine, or a mixture of sodium bicarbonate and HCO3- to keep the pH above 7.25, although the use of buffering agents is controversial. The fraction of inspired oxygen (FIO2) is adjusted to aim for an oxygen saturation of 85% to 95%. Protocols[1] guide the reversal of permissive hypercapnia in a similar fashion. PaCO2 levels are allowed to decrease slowly, changing from 10 to 20 mm Hg per hour when the PaCO2 is more than 80 mm Hg and changing even more slowly as levels reach the normal acceptable range.[1]

**Hypercapnic Acidosis**

Researchers are investigating whether hypercapnic acidosis itself protects the lung. It appears that hypercapnic acidosis protects the lung from injury due to free radicals and endotoxins. Hypercapnic acidosis lessens inflammatory responses by blunting lung neutrophil recruitment, cytokine concentration, cell apoptosis, and the production of free radicals. It seems that acidosis protects the body's organ systems. In animal studies,[3] hypercapnic acidosis reduced injury to the brain caused by hypoxia and ischemia. Other studies[3] have looked at the function of acidosis in protecting myocardial tissue and reducing liver damage. In light of this research, it may be that administering buffers to correct acidosis decreases this protection. Moreover, the use of HCO3- to correct acidosis has been questioned, as reflected by the decision to remove routine use of HCO3- from the cardiac arrest algorithm.[3] Still, researchers[3] are studying the impact of increasing levels of carbon dioxide while buffering acidity.

Hypercapnia can cause problems. Due to the vasodilating effect of carbon dioxide, permissive hypercapnia is contraindicated in patients with cerebral trauma, cerebral hemorrhage, and/or lesions in the cerebrum. In these patients, an increase in PaCO2 could increase intracranial pressure and cause more harm. Permissive hypercapnia is relatively contraindicated in patients who are hemodynamically unstable due to the tendency for it to decrease myocardial contractility, increase arrhythmias, and increase sympathetic activity.[1] Hypovolemia is another contraindication for permissive hypercapnia due to the tendency for acute increases in PaCO2 to cause a transient fall in cardiac contractile force, resulting in cardiovascular collapse.[6] There is also a concern that using permissive hypercapnia in combination with high FIO2 may lead to resorption atelectasis over time.[7]

**ARDS and Status Asthmaticus**

A landmark study[8] published in 2000 gave convincing proof that supported protecting the lung using a strategy of hypoventilation and minimal peak pressure. The study showed reduced mortality when lower VTs (about 6 mL per kg of predicted weight) and pressures (with a mean of 25± 6 cm H20) were delivered. Predicted weight (kg) was determined by subtracting 152.4 from height (cm), multiplying the result by 0.91, and adding 50 for males or 45.5 for females. The investigators used high respiratory rates and HCO3- infusion to correct or minimize elevated PaCO2 and acidosis encountered in patients receiving the lower VT.

A 2002 study[9] examined the use of low-volume, pressure-limited ventilation (LVPLV) along with permissive hypercapnia, compared with conventional mechanical ventilation, in septic and nonseptic patients with ARDS to evaluate the level of shunting. The study concluded that in ARDS, LVPLV with permissive hypercapnia tended to increase shunting. The researchers noted that PaO2 values did not drop due to increased venous oxygen (PvO2) levels. The higher PvO2 levels were the result of increased cardiac output due to stimulation by the high PaCO2.

A high PaCO2 increases pulmonary vascular resistance through its effect on pH; as pH drops, pulmonary vasoconstriction occurs, particularly if hypoxemia is present.[10] In light of this, a recent publication[11] discussing mechanical ventilation in patients with ARDS mentioned the use of nitric oxide to reduce both pulmonary hypertension and the potential for worsening pulmonary edema resulting from permissive hypercapnia.

The maximum limits for pH and PaCO2 have not been established. Two extreme cases of permissive hypercapnia related to ventilating patients in status asthmaticus were reported6 in 2002. The patients, both female, were 24 and 28 years old. They were treated using mechanical ventilation, permissive hypercapnia, and inhaled anesthetics. Both patients spent several hours using a ventilator, while sustaining PaCO2 levels of more than 150 mm Hg and pH levels of less than 7. The first case had a PaCO2 documented at 202 mm Hg and a pH of 6.68, and the second case had a PaCO2 of 218 mm Hg and a pH of 6.9. Mechanical ventilation of status asthmaticus patients involves avoiding dynamic hyperinflation, which is caused by prolonged expiration and premature airway closure. Use of small VTs and permissive hypercapnia in these patients appears to be useful in avoiding dynamic hyperinflation.

**Conclusion**

Permissive hypercapnia has been proposed as a strategy to protect the lung, and may even bring about an additional degree of protection by preventing the cell-mediated responses to overdistension that damage lung tissue. Permissive hypercapnia has been studied in the context of ventilating patients with ARDS, status asthmaticus, and COPD, and has a place in the arsenal of ventilatory choices. Using permissive hypercapnia carries some risk and has clear contraindications, and appears to be a choice held in reserve; some of the other tools used to manage difficult patients appear to be more effective and less troublesome. Still, clinicians need to understand the concept, implications, and potential uses of permissive hypercapnia because it is impossible to know when it might be the right approach to a complicated case.

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